

Response to Comment on “Reelin Promotes Peripheral Synapse Elimination and Maturation”

As the senior author of Quattrocchi *et al.* (1), I have attempted to reproduce the major findings described in this report in my laboratory, and to reconcile them with the findings of Bidoia *et al.* (2). Toward this end, I have reanalyzed the neuromuscular junction in the original *reeler* strain used for our study, and provided the mice to Misgeld, Lichtman, and Sanes [St. Louis, MO (2)] to rule out possible differences in genetic background or technique.

My findings agree with those of Bidoia *et al.* (2). Consistent with their observations, I saw no evidence of supernumerary synapses in *reeler* muscle fibers. I also did not observe the reported high frequency of polyneuronal innervation of *reeler* end plates, despite the

use of approaches and conditions similar to those used by Quattrocchi in (1). Thus, our published report indicating the involvement of Reelin in synapse elimination clearly appears to be in error. This raises serious doubts concerning the ability of exogenous Reelin to promote axonal withdrawal in a manner dependent on its reported serine protease activity (1, 3).

Although I was able to confirm the presence of small and poorly developed end plates in our inbred *reeler* mice (1), I agree with Bidoia *et al.* (2) that this phenomenon may result from muscle atrophy in these severely affected mutant mice—and not by a failure in the refinement of synaptic connectivity. The immaturity of *reeler* end

plates may be influenced by the genetic background and by the severity of the phenotype, as I did not observe it in the *reeler*-Orleans strain (provided by A. M. Goffinet, University of Louvain, Belgium), another *Reelin* mutant strain.

In conclusion, the results of my inquiry indicate that contrary to our original report (1), Reelin does not regulate the development of the neuromuscular junction. A retraction has been published (4).

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